Polyclonal Anti-Mitofusin 2, *MFN2 (*Sepharose Bead Conjugate)

Catalogue No. PA1051-S

Lot No. 09G02

Ig type: rabbit IgG

Size: 100µg/vial

Specificity

Human, mouse, rat. No cross reactivity with other proteins.

Recommended application

(Immunoprecipitation(IP)

Immunogen

A synthetic peptide corresponding to a sequence mapping at the N-terminal of human MFN2, different from the related rat and mouse sequence by single amino acids.

Purification

Immunogen affinity purified.

Formulation

50% slurry in PBS pH 7.2 with 0.01mg NaN_3a_3 preservative.

Storage

Store at 4˚C for frequent use.

Description:

This Antagene antibody is immobilized via covalent binding of primary amino groups to N-hydroxysuccinimide (NHS)-activated sepharose beads. It is useful for immunoprecipitation assays

BACKGROUND

Mitofusin 2 (MFN2) is a mitochondrial transmembrane GTPase regulating mitochondrial fusion and that the nucleotide-dependent activation of MFN2 concomitantly protects the organelle from permeability transition. It is mapped to chromosome 1 and encodes a 757-amino acid protein that contains an ATP/GTP-binding site motif. It is expressed in many tissues and cell lines such as brain and KG-1 with the highest expression in heart and skeletal muscle. This protein contains an N-terminal GTPase domain and a transmembrane domain near the C terminus. It shares 60% identity with MFN1. When stably expressed in COS-7 cells, MFN2 colocalizes with mitochondrial markers. Axonal CMT type 2A and autosomal dominant HMSN VI are caused by MFN2 and mutations in MFN2, which emphasizes its important role of mitochondrial function for both optic atrophies and peripheral neuropathies. ***REFERENCE***

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3. Santel A, Fuller MT. Control of mitochondrial morphology by a human mitofusin. J Cell Sci. 2001 Mar; 114(Pt 5):867-74. 4. Zuchner S, De Jonghe P, Jordanova A, Claeys KG, Guergueltcheva V, Cherninkova S, Hamilton SR, Van Stavern G, Krajewski KM, Stajich J, Tournev I, Verhoeven K, Langerhorst CT, de Visser M, Baas F, Bird T, Timmerman V, Shy M, Vance JM. Axonal neuropathy with optic atrophy is caused by mutations in mitofusin 2. Ann Neurol. 2006 Feb; 59(2):276.

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